With the Author's
Compts

On the Physiological Action of Nitrous Oxide.



BY

DUDLEY WILMOT BUXTON, M.D.,
B.S.Lond., M.R.C.P.,

Administrator of Anæsthetics in University College Hospital, the Hospital for Women, Soho Square, and the Dental Hospital of London, &c.

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"Quant à l'action anesthésique en elle-même, elle est parfaitement independente de l'asphyxie, et elle se produit par un tout autre mécanisme."—CLAUDE BERNARD.

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MR. PRESIDENT AND GENTLEMEN,

In reintroducing to the notice of this Society the subject of nitrous oxide gas, I feel some preface is called for, some explanation demanded.

To most members of this Society it will not be difficult to recall the various occasions upon which the subject of nitrous oxide has been brought upon the carpet, and, for the time being, fairly threshed out. The Proceedings of the Odontological Society give evidence of two facts in relation to the subject of my paper: they reveal that the Society at large took a keen interest, an interest which I venture to think has in nowise flagged or grown less real as time has gone on; and then that the members of this Society are, as a rule, experts who speak from the standpoint of experience, and not from hearsay. I shall, with your permission, later on, have to cite the experience and experiments of members of this Society, and

shall amply substantiate the truth of my statement that the Odontological Society speaks as a body of experts.

I shall not detain you, gentlemen, with expressions of my own unfitness to address you upon a subject with which you are already familiar, because my fitness or unfitness will amply be shown before we part this evening. I will say that I, as a student of the great physiological puzzle, our body, am simply and earnestly seeking for truth. I come here to lay what facts I possess before you, and only desire to go away again with a fresh store of information gathered from my fellow workers in this field.

The subject before us, which is the physiological action of nitrous oxide gas, is one of wider compass than at first reveals itself. The literature devoted to it is abundant, but the results arrived at are not commensurate with the amount of ink spent and paper spoilt. This is not wonderful, since the question is one of extreme difficulty, experimental research undertaken in its elucidation is both arduous and laborious, and so many writers have adopted the easier plan of working up old material which they have garnished with crude criticism. The whole subject is at the present time really confused by conflicting theories and experimental results, which, while presenting a mass of information, are almost irreconcilable. This unsatisfactory

state of affairs has prompted me to undertake an attempt to clear up some points, and harmonise some discordant conclusions. The task before me is of course of vast magnitude, and certainly more onerous than I could hope to bring to a satisfactory termination were my temerity such as to make me contemplate the undertaking single-handed. As it is I contemplate a prolonged research, and was therefore most grateful when your courteous Secretary permitted me to lay the matter before you, so enabling me to enlist your sympathies in my work, and to submit to you certain conclusions and facts upon which I might seek the ripened criticism of your prolonged experience and careful personal observation. I confess that I, like Frankenstein, have not a few times shrunk in dread from the monster my hands and brain had fashioned. By a happy thought, however, and a second appeal to Mr. Woodhouse, I was able to quiet my fears. By cutting my monster in pieces he appeared less huge and cumbersome and altogether less formidable. In short I suggested, and my view was kindly fallen in with, that I might first be allowed to bring in review the facts which bear upon the physiological action of nitrous oxide gas, and to indicate how far the conclusions hold from the premises adduced, and how far they correspond with our present experience; while I should reserve for the present the more strictly experimental part

of my research until we had cleared the ground from mere *à priori* reasoning, and had sifted the evidence as at present placed in our hands.

Many of the facts which I propose to bring before you to-night will be familiar to you as facts, although I venture to think their true significance in many cases has been overlooked. This has occurred because there has not been an attempt made to bring them into sequence, and to trace the subtle cord of correlation which subsists between them. It is my aim to attempt so to marshal our facts that we may clearly ascertain their true bearing, and thus be enabled to see what gaps still exist in the chain of evidence.

Nitrous oxide—

(*a*) is said to produce a state which we term *anæsthesia*—a loss of sensation ;

(*b*) it initiates certain emotional states, provoking a sensation of exhilaration and well-being—in fact, it plays the *rôle* of a stimulant ;

(*c*) it gives rise to modifications of the respiratory and

(*d*) circulatory systems ;

(*e*) and provokes marked muscular movements, which may be roughly classed as

(i) rigidity or contracture, and

(ii) jactitations.

These are, speaking broadly, the effects of ni-

trous oxide upon the mammalian organism, and in attempting to explain them we must ascertain their relations among themselves. In the first place it is essential that we should assure ourselves whether the conditions of our problem are simple or complex. I propose to show that the latter is the case, and that many of the phenomena attributed to nitrous oxide are, in point of fact, merely accidents of our methods of introducing the gas into the lungs.

The main point of interest in the physiological action of nitrous oxide is the production of anæsthesia. Now this condition, the loss of power of perception of sensation, together with the state of analgesia, or loss of sensation of painful impressions, may arise from want of action of the sensory end-organs, of the conducting sensory nervous fibres, or of the receiving and perceptive centres. These distinctions the pathologist points to as resulting from various neuropathic lesions. We are all aware that local anæsthesia, as it is called, and local analgesia are determinable by such applications as aconitine, cocaine, cannabis indica, and we are aware that in certain rare cases etherisation will call into being a true analgesia, a condition in which a patient is spelled into a sensationless trance, while he possesses his faculties sufficiently to know what is going on.

That nitrous oxide is neither a true anæsthetic

nor a true analgesic is the first proposition I propose to consider. I submit that the evidence goes to show that sensation is retained until the perceptive powers themselves cease to receive. That sensations, whether merely tactile or painful, are not blocked at the end-organ, nor is their progress interfered with along the nerves which connect the sensory end-organs with the spinal cord and brain, and hence the stoppage is carried back to the centres themselves. A further fact is that I have observed, and I believe I am in harmony with most of the authorities upon the subject, that during the stage immediately anterior to the loss of consciousness, persons under the influence of nitrous oxide are *hyperæsthetic*. Now observe the significance of this fact. Under normal conditions, we move our limbs in response to stimuli, and do so in an orderly fashion; we experience sensations of heat or cold, of touch, light, and so on, without distress, and this is because the complex mechanism of our reflexes—the end-organ and conducting fibres to the nerve centres, the nerve centres themselves, and the efferent fibres thence to the muscles—are all acting harmoniously, and are under the control of the inhibitory centres in the cerebro-spinal axis. When from any cause these inhibitory centres are thrown out of gear we have on the one hand excessive muscular movements, on the other

excessive sensory impressions. A whisper becomes magnified to a roar, the fall of a pin assumes the importance of the crack of a whip, and so on. This, then, I venture to submit, shows that, if I am right in stating that nitrous oxide does induce hyperæthesia, the cerebro-spinal axis, at least as far as sensation goes, is the seat of the changes which nitrous oxide induces, and which culminate in the complete abeyance of consciousness. In investigating this fact I would, however, urge your attention to an important caution. The personal equation of sensibility varies within wide limits. So coarse are the senses in many persons—the lympho-phlegmatic—that hyperæsthesia in them scarcely amounts to more than the normal sensibility in the nervous temperament, and so we cannot with propriety reason from their cases. In this connection I would draw your notice to the convenience of grouping the brain phenomena due to nitrous oxide in three periods: the period before unconsciousness, which I contend is the hyperæsthetic period, the period of unconsciousness, and the period of returning consciousness, in which I believe nitrous oxide hallucinations take their origin.

Now if nitrous oxide does indeed effect molecular changes in the brain, a point to which we shall return later, we have yet to examine the means by which this gas enters the system and is enabled

to produce its central effects. We are here met by a most comprehensive problem. One of the following conditions presumably constitutes the mechanism for which we are seeking :—

(*a*) The nitrous oxide may either give rise to other bodies by changes in its own chemical form ; or

(*b*) Acting purely in a mechanical fashion it may upset the normal equilibrium of the function of respiration, and so give rise to accumulation of aërial fluids in the blood, and which would normally be excreted.

(*c*) It may act *per se* and exercise a specific action just as strychnine or any other body.

We will dispose of these propositions in the order in which they stand. At one period it was believed that nitrous oxide acted as an oxidising agent, by splitting up in the body or tissues into oxygen and a residuum of nitrogen compounds, and hence came into vogue the Apnœal or Hyperoxygenation theory of Colton. In support of this theory, Stillé and Maisch urge that venous blood is arterialised by shaking it with nitrous oxide, that phosphorus burns in it, that seeds germinate under bell-jars of nitrous oxide. Zimmermann, whose paper I have not been able to consult, is pledged by Stillé and Maisch to the statement that

pigeons and rabbits will recover after being placed in the gas for eight hours. Of course, were such contentions true, we should have to admit that nitrous oxide is a respirable gas. However, it is difficult to believe any of the assertions when viewed by the light of modern research. For it is well within the memory of this Society that the Committee appointed by it tendered, I believe in the year 1873, their second report, in which is given the results arrived at by Dr. Frankland. This observer came to the conclusion that nitrous oxide was not decomposed during its sojourn in the body, basing his opinion upon analyses made of air expired by rabbits when confined in an atmosphere of mixed air and nitrous oxide. With these analyses I will not detain you. Others, adopting less exact methods, have arrived at the same result, and Coleman pointed out that were oxygen liberated, it would probably produce the most baneful results upon the organism. However, this objection has little weight when we consider that it would be the tissues of the body which would take up the oxygen liberated from the gas and employ it for molecular integration. Jolyet and Blanche, who publish their results in Brown-Séquard's "Archives de Physiologie," find vegetables as well as animals die incontinently when placed in an atmosphere of nitrous oxide. And I have in the case of certain seeds failed to induce germi-

nation in an atmosphere of pure nitrous oxide. Of course combustion will take place in nitrous oxide, provided the heat be sufficient to produce "the mode of motion" in the molecules of the gas which leads to their disintegration; but experiment has shown that the heat of the blood is insufficient to initiate such a dissociation.

But perhaps the most crucial argument adducible in confutation of the hyperoxygenation theory is that apnœal states are by no means necessarily anæsthetic conditions. Bonwill's suggestion of rapid breathing to produce anæsthesia probably acts rather through the changes it produces in the blood pressure of the cerebral circulation or nervous exhaustion, than by dint of hyperoxygenation. But all these observers have apparently overlooked another way in which the nitrous oxide may be introduced into the system, and exist as a quasi-compound in the blood and respiring tissues. We are well aware that carbonic monoxide, hydrocyanic acid, and the nitrites, bodies of especial interest to us at present, are capable of entering into a combination with the colouring matter of the blood, oxyhæmoglobin, and that of the respiring tissues, oxy-myo-hæmatin, and forming more or less stable bodies which are isomorphous and present kindred physical properties. We cannot fail to recognise at least strong chemical bonds of union between nitrous

oxide and the nitrites, nor does it appear to me to be a far-fetched hypothesis to suppose that nitrous oxide enters the blood in some such a combination. At the same time I have no actual proofs of what I have suggested, since there is extreme difficulty in eliminating mere asphyxial from nitrous oxide spectrum if such a one exists. Of course when an animal is killed by nitrous oxide, the blood, if examined within two minutes of death, gives the well-known spectrum of reduced hæmoglobin, while the muscles give the corresponding one of reduced myo-hæmatin. Here we are dealing, not with nitrous oxide blood effects, but these complicated and probably overridden by asphyxial effects. Later on we shall have to point out a further possible way by which nitrous oxide may be introduced into the organism.

Let us now consider whether nitrous oxide can act simply mechanically by ousting the oxygen from the oxyhæmoglobin and oxy-myo-hæmatin, *in fine* as an indifferent irrespirable gas.

It is conceivable nitrous oxide may act as a simple irrespirable gas in precisely the same way as nitrogen, hydrogen, and so on, or it may deoxidise the blood acting as a reducing agent, such as ammonium sulphide ; in either case the end arrived at is the same, viz., asphyxia. It was to this conclusion that Jolyet and Blanche arrived when they wrote, "Le protoxyde d'azote étant un gaz irrespirable et ne

possédant les propriétés anesthesiques qu'on lui a attribuées, son emploi ne peut être que dangereux, et doit à ce titre être proscrit de la pratique médicale."

It has been said by those who regard nitrous oxide as analogous in its action to nitrogen, or any other indifferent gas, that it differs only in so far that one gas is able to oust oxygen from oxyhæmo-globin sooner than another, an assumption which seems to me to be purely gratuitous, since the reduction must take place in the tissues, the blood supplying them until its store of oxygen is exhausted, when asphyxia takes place. Bernard, whose classical work on asphyxia gives the fullest account of that condition with which I am acquainted, speaks of three forms of asphyxia:

(1) That arising from inhalation of irrespirable gases which are themselves harmless. To this class many authorities would relegate nitrous oxide.

(2) That due to poisonous vapours, such as carbonic monoxide, sulphuretted hydrogen; and

(3) Asphyxia from want of air, such, for example, as would ensue upon ligature of the trachea. He subsequently points out, and I think with justice, that asphyxia resulting from the inhalation of an indifferent irrespirable gas, and that due

to want of access to air, are in fact one and the same condition.

Let us now consider very carefully indeed the process of phenomena ensuing upon deprivation of oxygen, that we may compare them with the course of events in nitrous oxide narcosis. It seems to me that, even if we fail to establish what is the exact nature of nitrous oxide narcosis, we cannot fail to see many striking discrepancies between it and asphyxia pure and simple.

The arterial blood becoming gradually deoxidised, by parting with its oxygen to the tissues, acts upon the Medulla respiratory centres; hence follows hyperpnœa. This in the case of some persons subjected to experiments by Drs. Burdon Sanderson, John Murray, and Mr. J. Smith-Turner, who caused them to respire pure nitrogen, did not occur for about two minutes after commencement of inhalation. With the increased venosity of the blood follows the tumultuous breathing of dyspnœa when all the accessory muscles are called into action. Subsequently expiration becomes markedly in excess of inspiration. Expiratory convulsions then ensue. The convulsions cease owing to the exhaustion of the nervous centres, the pupils are dilated, the conjunctival reflex cannot be elicited, the muscles are flaccid and quiet. At ever lengthening intervals the respiratory centre gathers itself together, and starts a long

inspiration; these grow more and more shallow, and at length cease. Lastly the heart stops. It is convenient to separate these phenomena into three stages:

(1) Dyspnœa, when both inspiration and expiration are in excess;

(2) Convulsions, when expiratory efforts exceed inspiratory;

(3) Exhaustion.

Now let us contemplate the circulatory changes occurring during the procession of these phenomena. The first change of importance is a rapid rise of blood pressure; this, persisting during the first and second stages, is due to stimulation of the vaso-motor centres by the venous blood, resulting in a constriction of the arterioles. Coincident with this is an acceleration of the heart beat; this, however, rapidly gives place to slowing and increase in the force of the beat. During the third stage the blood pressure rapidly falls to nil, and the heart beats become very slow and feeble, and finally cease. The duration of these stages of asphyxia have not been accurately determined in the case of human beings. In dogs the first stage occupies about a minute, and death ensues in four or five minutes. In the cases cited above, when patients were partly asphyxiated with nitrogen, they were subjected to the process for periods varying from three minutes to four minutes, and

yet had evinced none of the phenomena usually found in the second stage of asphyxia. We also know that voluntary breath-holding can be maintained for over a minute; hence it would seem not improbable that man takes at least as long as dogs to die from deprivation of oxygen.

Now I would ask your close attention to the phenomena of the so-called asphyxia of nitrous oxide narcosis.

In the first place we find a tolerably uniform increase in the number and depth of respirations. I have never as yet succeeded in detecting any excess of expiratory over inspiratory movements. The respirations are, and I speak under correction, simply an exaggeration of the normal, quite regular, but hurried in rhythm and increased in depth. And this begins certainly within half a minute, usually within fifteen seconds, from the commencement of nitrous oxide inhalation. The respirations, however, slow as narcosis proceeds, and finally stertor supervenes, which frequently is followed by a period of complete respiratory calm, no thoracic movements appearing. A few seconds more and respirations are recommenced, and the person passes quietly into the ordinary swing of his breathing. I have never observed anything which in the slightest degree resembled the expiratory convulsions one is so familiar with in the case of the lower animals killed by asphyxia.

Again, in considering this subject it occurred to me that one should find the same circulatory changes taking place during nitrous oxide narcosis as obtain during asphyxia were the conditions one and the same. I am at present engaged upon this research, but have not been able to place the matter beyond doubt. In a series of sphygmographic traces, some of which I have placed before the Society, I have sought to determine what variation in tension occurs during nitrous oxide narcosis. These traces were taken upon persons sufficiently accustomed to inhaling the gas to eliminate as far as possible the circulatory changes due to fear, &c. I need hardly add no surgical procedure was undertaken, as such must have introduced the disturbing element of shock. I mention this as in some of the traces published such surgical procedure has been allowed, and so, as I think, a most disturbing factor introduced. In conducting these experiments it has been my custom to take a tracing of the normal pulse before narcosis was commenced; and then after ten seconds or so of gas another trace was taken, and this was continued until consciousness returned.

In no case have I succeeded in obtaining any marked increase in tension in the pulse trace, and there has appeared to me to be an actual lessening of the tension during narcosis, as evidenced in the loss of tidal wave. I should mention that

in most cases the narcosis was pushed very far, jactitations and extreme cyanosis being induced. A careful study of the tracings now before the Society* will reveal the following facts. In the first place there is a very notable acceleration of the pulse, and this persists throughout the whole period of narcosis; secondly, the following signs of a lowered tension will be recognised. The tidal wave is lessened or almost lost, the dicrotism of the pulse becomes strongly marked; the acuteness also of the initial curve will be seen to be considerably increased. A further point will be observed in some of the traces, viz., that the dicrotic wave is further down the curve when the patient is under the influence of the gas than is normal. These evidences of lowered tension appear to me to be constant in my results, although of course I can only show a few specimens here to-night. Now, unless we deny the evidence before us, it seems at least remarkable that nitrous oxide should cause such evidence of lowered tension, unless it was really a fact that in some way nitrous oxide is capable of lowering the tension of the blood in the arteries, and of accelerating the heart's action. At the same time I am anxious not to seem to attach an undue importance to sphygmographic records; I would simply urge that taken with the utmost caution they reveal a fairly uniform result. To

* See pp. 155–158.

investigate this question of blood pressure still further I have undertaken some experiments whereby the actual pressure in the blood vessels of dogs has been determined. The results of these I hope to lay before you upon a subsequent occasion, but I may say that when due allowances are made I believe the results will go to strengthen my present contention, that nitrous oxide pushed to the extent of narcosis does not give rise to circulatory changes at all comparable to those occurring in the course of asphyxia.

The results obtained by the cardiograph in the human subject have not been encouraging, and I have therefore determined to investigate the exposed hearts of mammals as likely to render a more reliable record. In this connection the experiments of Amory, Krishaber, Goldstein, and Kuntz are of interest. These observers found that animals, when subjected to the vapour of nitrous oxide, after a time died. Now it is important to notice that the animals, when made the subject of a post-mortem examination, revealed the usual signs of asphyxial poisoning. But this is of course a wholly different thing to death from nitrous oxide. In these animals nitrous oxide narcosis was followed by suffocation, air was excluded, and hence asphyxia ensued. I believe it has happened to skilled anæsthetists to have a necessity of maintaining nitrous oxide narcosis for

a considerable time. This has been done by allowing the patient to respire air at long intervals. In this way a really prolonged narcosis can be effected. Clearly in such cases the narcosis was something wholly outside of the concurrent asphyxia, and it is the great disadvantage of our present methods of administering nitrous oxide that we can hardly avoid producing more or less asphyxia while we are seeking only to promote the action of nitrous oxide upon the nervous centres. M. Paul Bert has by his well-known plan sought to obviate this drawback. I do not propose to address you upon his method, as that is as well known to you as to me, but I claim your attention to the important fact which underlies the success of M. Paul Bert's system. It is, that so long as he succeeds in supplying the bodily need of oxygen side by side with enough nitrous oxide to induce narcosis, he is able to achieve a prolonged unconsciousness, and yet his patient maintains his respiratory and vital equilibrium. I have no personal experience of the method, nor am I aware whether a narcosis lasting so long as an hour has been achieved.

It yet remains to be shown whether nitrous oxide acts in virtue of a power it might possess of reducing the oxyhæmoglobin of the blood, the oxy-myo-hæmatin and oxyhæmoglobin of the tissues. Were this the case we should expect to find it capable of either fixing the oxygen, as

in the case of carbonic monoxide hæmoglobin, or of producing a very rapid asphyxia, which I have attempted to show it does not do. I am also unaware of any facts which show that nitrous oxide is capable when in the blood of linking itself, on the one hand, with another molecule of oxygen, or on the other of fixing the oxygen in any other tissues, including the blood; if neither of such occurrences actually happen, it is hard to see what becomes of the oxygen, even were nitrous oxide capable of ousting it. As I have pointed out above, the evidence we derive from spectroscopic examination is not convincing, as MacMunn only examined the blood after death, and not simply during narcosis. A test of great value would be the examination of blood shed during an operation performed when narcosis after Paul Bert's method had been obtained.

We may, I think, then, conclude that nitrous oxide produces narcosis by virtue of other than asphyxiating qualities.

This gas, then, enters the blood through the lungs and exercises a certain specific action upon the nervous centres. And here we have a ready explanation of an otherwise most anomalous circumstance, viz., that in a certain number of cases persons evince the utmost toleration for nitrous oxide, and resist its nepenthal action for a minute or more. I doubt not members who are here

to-night can furnish me with instances of this idiosyncracy. In other words, these persons need a larger *dose* of this agent, nitrous oxide. It is inconceivable, I think, that were the effect other than a specific one, that quantity, or dose, as I prefer to call it, could have any effect. To explain. Were I to curarise a rabbit, I should produce complete paralysis of all its muscles, and the creature would die asphyxiated. Now I should always produce this result whatever quantity above a certain dose was given; the asphyxia is mechanical. So, were nitrous oxide purely an asphyxiant, I should invariably narcotise my patient when I had replaced his oxygen; but such is not the case—in fact, I narcotise him *before this oxygen is exhausted*, and I submit a long while before.

But there yet remains to discuss in what consists this specific action of nitrous oxide. As I explained, it is impossible for me in the space at my disposal to enter at all fully upon this question upon the present occasion, and so I will confine myself to a brief summary and leave it for elaboration in my next paper.

Nitrous oxide, then, may be taken as in all probability entering the blood by forming a loose combination with either the oxyhæmoglobin or the globulins and other albuminous bodies. It then passes to the nerve centres, and gives rise to subjective exhilaration, acting in fact as a

stimulant. Upon the heart it would in this stage appear to act mainly as an accelerator, but in what way I am at present not prepared to hazard a speculation. Subsequently the nervous centres pass from their initial stimulation to a condition of narcosis in which volitional activity is in abeyance. At this time the inhibitory centres are in a state of at least lessened activity, as is evidenced by involuntary movements, micturition, &c.; later the reflexes are lost. During this stage the blood pressure would appear to be lessened, the action of the heart accelerated, and the respiratory rhythm, at first quickened, subsequently slowed to a stand-still. This state of things persists for awhile, and is accompanied by relaxation of some muscles, *e.g.*, the palatine and faucial muscles, while other muscles are the seat of arhythmic clonic and tonic contractions. Following upon this stage, we usually meet with the phenomena of recovery. At the present time I shall restrict my attention to these, because when the gas is pushed further the resulting action is a complex one, and largely due to want of access of oxygen. During the period of recovery, a further stage of excitement appears, and it is commonly associated with hallucinations, sometimes pleasant, sometimes extravagant. The senses soon become keenly upon the alert, and operative measures prolonged into this period give rise to the most intense

pain. Patients will declare the pain in such cases transcends that where no gas is given.

Here, then, is very briefly summarised a theory, at present inadequate and incomplete, of the action of nitrous oxide; to it I shall hope to return and to offer some further elaboration of a subject as difficult as it interesting, as complex as it is important.



FIG. I.—Normal pulse, H.B.

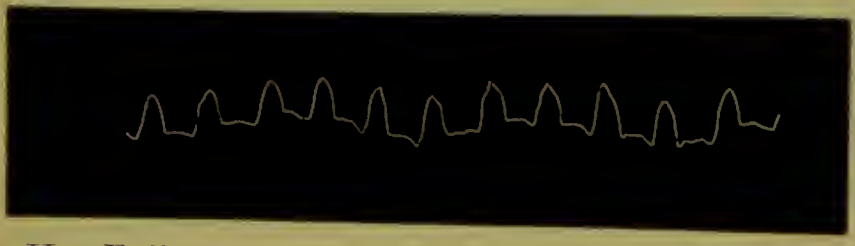


FIG. II.—Fully under nitrous oxide; shows no tidal wave; marked dicrotism, evidencing lowering of arterial tension.



FIG. III.—Shows gradual resumption of normal characters during "coming to."



FIG. IV.—Continuation of III; tidal wave appearing.

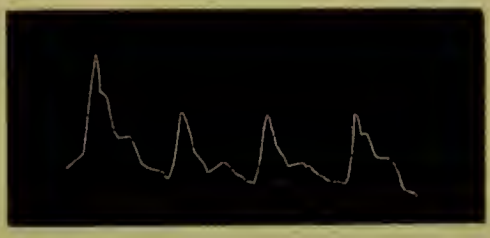


FIG. A. I.—Normal pulse.

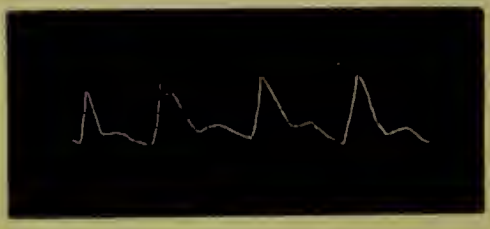


FIG. A. II.—Under gas, (*a*) Shows marked acceleration of rate of heart beat; (*b*) also loss of tidal wave, evidencing lowering of tension.

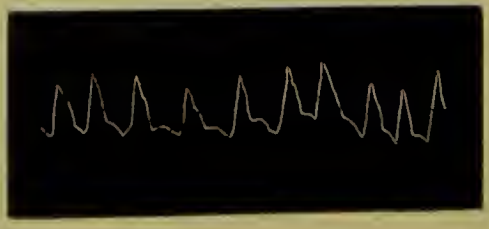


FIG. A. III.—This trace shows great acceleration of rate of heart beats, absence of tidal wave, acuteness of apices of curves, markedly increased respiration curves.



FIG. B. I.—Normal trace, H.S.

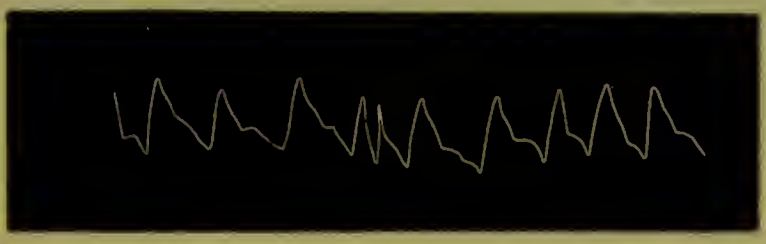


FIG. B. II.—Under—shows loss of tidal wave; pointing of apex; respiratory curves exaggerated; shows lowered tension.

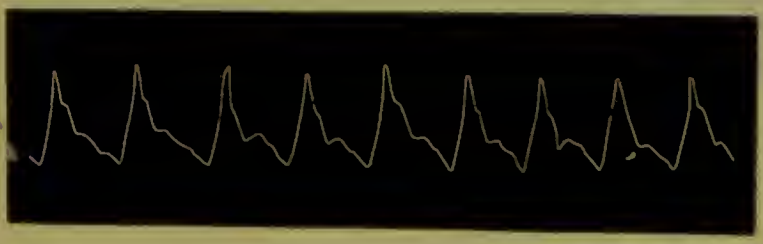


FIG. B. III.—Still under, beginning to come to.

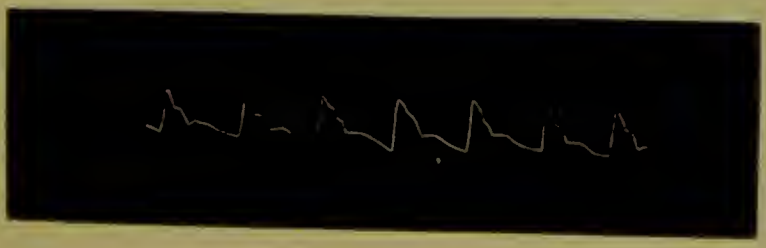


FIG. B. IV.—Resumption of normal beat.

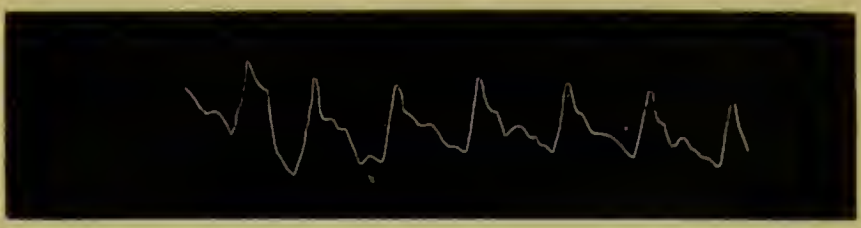


FIG. C. I.—Normal beat, shows tidal wave, aortic notch, dicrotic wave.



FIG. C. II.—Under nitrous oxide. Shows acceleration of heart beats; increased acuteness of curve; diminution of tidal wave; dicrotic wave occurs later, this is shown by being farther from apex.



FIG. C. III.—Nearly same as II, but features more marked.

HARRISON AND SONS, PRINTERS IN ORDINARY TO HER MAJESTY, ST. MARTIN'S LANE.